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METHODS

Novel Approach to the Analysis of Restenosis After the Use of Three New Coronary Devices

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Restenosis after coronary intervention has remained a vexing problem despite the introduction of nearly 24 newer coronary interventional devices. To more clearly evaluate the potential impact of three such new devices on restenosis, coronary lumen diameters were measured before, immediately after and at 6 months after intervention, and restenosis was analyzed using continuous geometric techniques.

Lumen diameters were measured before and immediately after intervention in 223 coronary vessels treated with one of three new devices: a single Palmaz-Schatz stent (n = 87), directional atherectomy (n = 125) and laser balloon angioplasty (n = 11); 184 (83%) of the patients underwent follow-up angiography 6 months after treatment. The immediate increase in lumen diameter produced by the intervention (acute gain) and the subsequent reduction in lumen diameter between the time of intervention to 6 month follow-up study (late loss) were examined. For each of the three interventions, the restenosis rate at follow-up study was analyzed using a traditional dichotomous definition (≥50% diameter stenosis), as well as a novel graphic technique.

Although the apparent restenosis rates differed significantly among the three interventions (19% for stents, 31% for atherectomy and 50% for laser balloon angioplasty; p = 0.02), late loss among the three interventions was equivalent (average 1 mm; p = 0.91). There were, however, marked differences in the acute gain achieved by the three interventions: 2.6 mm for stents, 2.2 mm for atherectomy and 2 mm for laser balloon angioplasty; p < 0.001). It was these differences in acute gain rather than late loss that explained the observed differences in restenosis rate.

By using a continuous rather than a dichotomous view of restenosis and resolving long-term device results in the components of acute gain and late loss, analysis of this patient set provides unique insights into the potential role of these new devices in reducing restenosis. The absolute magnitude of late loss in lumen diameter (reflecting local intimal hyperplasia) was equivalent among the three devices and was greater than the late loss previously reported for conventional balloon angioplasty. Therefore, the ability of a newer device to reduce restenosis may be related more to its ability to provide the greatest acute gain in lumen diameter, rather than the ability to reduce subsequent intimal hyperplasia.

(J Am Coll Cardiol 1992;19:1493–9)

Conventional percutaneous transluminal coronary angioplasty has continued to be troubled by an approximately 30% incidence of restenosis within 6 months of an initially successful procedure (1–3). To address this and other limitations of conventional angioplasty, nearly 24 newer devices for coronary intervention (including stents, atherectomy catheters, laser balloon angioplasty catheters and ablative laser catheters) are now in active clinical evaluation (4–6). We treated >200 patients with one of three such devices during the period from June 1988 through August 1990. On the basis of 6-month angiographic follow-up study in 83% of these patients, we sought to address two major questions. 1) Is there any evidence that these devices have a lower incidence of restenosis than that expected from conventional angioplasty? 2) If so, what are the apparent mechanisms that underlie the apparent interdevice differences in observed restenosis rates.

Methods

Study patients. All patients were enrolled in a device-specific clinical protocol supervised by the Committee on Clinical Investigation at Beth Israel Hospital. They were selected for treatment with one of the three investigational catheters on the basis of presence of symptomatic myocardial ischemia and the presence of restenosis after one or more prior interventions (108 lesions, 48%) or lesion characteristics (for example, eccentric, ostial or saphenous vein graft lesions) thought to be unfavorable for conventional dilation (143 lesions, 64%) (Table 1). These new device

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Manuscript received September 28, 1990; revised manuscript received October 10, 1991, accepted January 6, 1992.

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0735-1097/92/$5.00

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Table 1. Patient and Lesion Demographic and Angiographic Characteristics

	Stents	Atherectomy	Laser Balloon Angioplasty
Lesions (no.)	87	125	11
Patient age (yr)	58 ± 11	57 ± 11	61 ± 9
% Male	83	83	82
Vessel treated (%)			
LAD	18	66	55
RCA	47	13	36
LCx	6	7	9
SVG	29	14	0
Prior restenosis (%)	62	43	0
Lesion morphology (%)			
Eccentric	29	72	64
Ostial	0	9	0
Device success (%)	99	92	100
Lumen diameter (mm)			
Baseline			
Reference	3.35 ± 0.31	3.09 ± 0.69	2.78 ± 1.89
Lesion	0.81 ± 0.6	0.68 ± 0.38	0.45 ± 0.25
% Stenosis	77 ± 16	78 ± 11	84 ± 9
Lesion length	6.95 ± 3.31	6.61 ± 3.3	3.82 ± 1.89
Postprocedure			
Lesion	3.38 ± 0.62	2.90 ± 0.57	2.43 ± 0.29
% Stenosis	−1% ± 13	6% ± 15	15% ± 10
Acute gain	2.57 ± 0.77	2.22 ± 0.63	1.98 ± 0.42
Follow-up			
Lesion	2.35 ± 0.92	1.86 ± 0.96	1.46 ± 0.82
% Stenosis	29 ± 25	37 ± 29	50 ± 26
Late loss	1.08 ± 1.06	1.02 ± 0.96	0.99 ± 0.76

LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery; SVG = saphenous vein graft.

procedures accounted for 14% of the 1,593 coronary interventions performed during the study period.

Coronary stent group. These patients were treated with the Palmaz-Schatz balloon-expandable stent (Johnson & Johnson Interventional Systems) as previously described (7). In brief, the target lesion was crossed with a 0.014-in. (0.036-cm) guide wire and predilated with a conventional 3-mm balloon. The balloon catheter was then exchanged for a balloon-mounted stent advanced through a protective delivery sheath (except for the first 38 patients who were treated with a sheathless delivery balloon [7]). The stent was positioned and deployed by balloon inflation and expanded further with a larger balloon (up to 5 mm) as needed to match stent diameter to that of the adjacent reference vessel segment. The eight patients treated with multiple overlapping (rather than single) stents early in our experience were excluded from further analysis given the known twofold higher incidence of restenosis at the stent overlap site (7). Stent placement was performed during infusion of dextran-40 and systemic heparinization (activated clotting time ≥300 s), with concomitant administration of aspirin (325 mg/day) and dipyridamole (200 mg/day). Heparin infusion was continued until administration of warfarin (5 to 10 mg/day) increased the prothrombin time to 16 to 18 s, at which level it was maintained for 6 to 12 weeks to minimize the risk of stent thrombosis before full endothelial covering. None of the patients so treated in this study sustained acute or subacute vessel thrombosis, as has been reported (8) in up to 24% of patients receiving another type of coronary stent.

Directional atherectomy group. These patients were treated with the Simpson AtheroCath (Devices for Vascular Intervention) as previously described (9). In brief, the target lesion was crossed with a 0.014-in. (0.036-cm) guide wire and an appropriately sized atherectomy catheter (6F for vessels ≤3 mm and 7F for vessels between 3 and 4 mm in diameter) was advanced across the lesion. Three to 15 passes were performed until the desired lumen diameter was achieved or no further plaque could be retrieved. The procedure was performed under systemic heparinization, with concomitant aspirin and dipyridamole therapy, as described for the coronary stent group.

Laser balloon angioplasty group. These patients were treated with the Spears laser balloon catheter (USCI) (10). After predilation with a conventional balloon, the laser balloon (inflated diameter 3 to 3.5 mm) was positioned and inflated to 60 psi as previously described (10). After delivery of 15 to 30 W for 20 s, the artery was allowed to "cool down" for 30 s, at which time the balloon catheter was deflated and removed. Ten patients treated on an emergency basis with the laser balloon for the reversal of abrupt closure caused by conventional angioplasty were excluded.

Acute and short-term outcome analysis. Criteria for device success included passage of the device, a residual diameter stenosis ≤50% and the absence of a major complication (death, Q wave myocardial infarction or emergency bypass surgery). Only two patients sustained any of these major complications (one patient after atherectomy and one after an unsuccessful attempted stent placement required emergency surgery). Successfully treated patients were encouraged to undergo serial exercise testing at 1, 3 and 6 months after the procedure and repeat coronary angiography at 6 months or if indicated clinically. However, angiographic studies that did not show restenosis (diameter stenosis ≥50%) were included only if performed >4 months after the index procedure. Results from the first 37 patients who received a stent (7), the first 76 patients who underwent atherectomy (9) and the 11 patients who underwent laser balloon angioplasty (10) have been reported previously.

Both the acute (pre- and postprocedure) and 6-month (follow-up) angiograms were analyzed by using optically magnified images. The minimal lumen diameter of each treated lesion was measured by using the known diameter of the angiographic catheter (11,12). The reference size was taken as the mean of the diameter of the vessel segments proximal and distal to the treated lesion. On the basis of these measurements, two important variables were derived. *Acute gain* was defined as the increase in the absolute diameter of the treated segment immediately after the procedure. *Late loss* was defined as the decrease in absolute diameter of the treated segment from the postprocedure to

the 6-month follow-up angiogram. The long-term net gain produced by an intervention is thus the difference between its acute gain and late loss.

Statistics. Clinical data and the coronary dimensions were reported as mean value ± SD. Immediate and follow-up results were compared by using analysis of variance. A p value <0.05 was considered significant. The distribution of late loss for the entire group was tested for normality by the Kolmogorov-Smirnov test; distributions were displayed by histogram.

Results

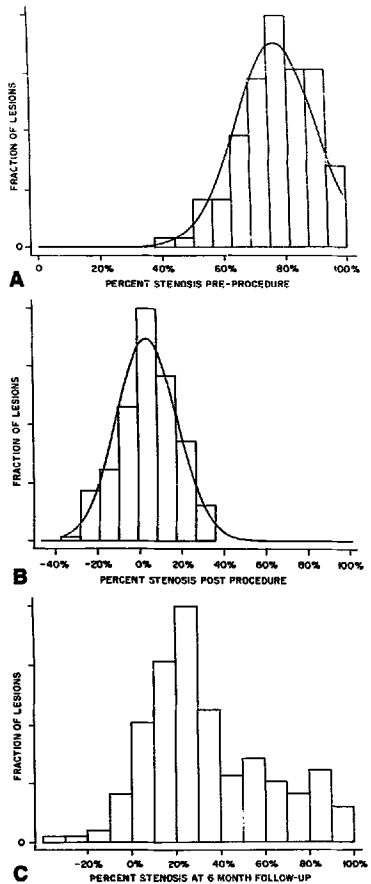
Device success. Device success was obtained in 95% of 235 lesions: 87 (99%) of 88 stents, 125 (92%) of 136 atherectomy procedures and 11 (100%) of 11 laser balloon procedures. Six-month angiographic follow-up data were available in 184 (83%) of the 223 successfully treated vessels (90% of stent, 77% of atherectomy and 100% of laser balloon angioplasty lesions). The distributions of percent diameter stenosis before and after treatment and at the time of follow-up angiography in each group are shown in Figure 1.

These follow-up data may also be displayed as a cumulative frequency distribution, normalized to 100% of each type of lesion to correct for different numbers of lesions treated (Fig. 2A). This plot shows the overall rate of restenosis (defined as late diameter stenosis ≥50%) to be 27%. Restenosis rates for the individual devices, however, varied significantly (19% for stents, 31% for directional atherectomy and 50% for laser balloon angioplasty; $p = 0.02$) as reflected by the disparate cumulative frequency distribution plots for the different devices (Fig. 2B).

Acute gain and late loss for each device. Because late lumen diameter (and restenosis) are dependent on both the increase in lumen diameter immediately after the procedure and the magnitude of intimal response over the ensuing months, we then examined data pertaining to the acute gain and late loss for each device (Table 1, Fig. 3). Stenting produced the greatest acute gain of 2.6 mm (increase in lumen diameter from 0.8 to 3.4 mm, reference diameter 3.4 mm), with a subsequent late loss of 1 mm (decrease from 3.4 to 2.4 mm). Atherectomy produced an acute gain of 2.2 mm (increase from 0.7 to 2.9 mm, reference diameter 3.1 mm), with a subsequent late loss of 1 mm (decrease from 2.9 to 1.9 mm). Laser balloon angioplasty produced the smallest acute gain of 2 mm (increase from 0.5 to 2.4 mm, reference diameter 2.8 mm), with a late loss of 1 mm (decrease from 2.4 to 1.5 mm). Although there were significant ($p < 0.001$) differences in acute gain, late loss was equivalent ($p = 0.91$) among the three devices.

Given the identical average late losses among the three devices, a histogram of late loss for all lesions was constructed (Fig. 4), demonstrating that late loss is continuously and normally distributed ($p = 0.99$, probability of nonnormal distribution). The potential differences in late loss based on the specific device used, reference artery size, acute gain,

Figure 1. Histograms demonstrating the normal distribution of percent stenosis before (A) and after (B) intervention (stenting, atherectomy and laser balloon angioplasty) in 223 lesions and in 184 lesions at 6-month follow-up after intervention (C). The distribution in C is broader and more rightwardly skewed compared with that in B.



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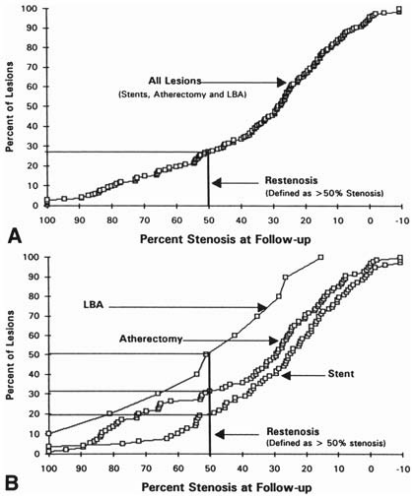

Figure 2. Definition-independent view of restenosis. **A,** Percent stenosis at 6-month follow-up study from Figure 1C is displayed as a cumulative distribution of all lesions. Starting at the most severe (100%) stenoses, the value on the ordinate represents the percent of lesions with a stenosis at follow-up angiography that is greater than the corresponding value on the abscissa. With this graph, any definition of percent stenosis can be selected to calculate the corresponding percent of the study group that would qualify for restenosis by that definition. If a >50% stenosis definition is used, 27% of the study group is seen to have restenosis. **B,** Display of definition-independent restenosis curves for the individual devices. Significant differences in restenosis rates (here defined as >50% stenosis) are evident (50% for laser balloon angioplasty [LBA], 31% for atherectomy and 19% for stents), but the uniform differences among the three devices in their cumulative stenosis distributions at 6-month follow-up study are also evident.

lesion morphologic characteristics, patient demographics and prior restenosis could not be evaluated further because of inadequate sample size for multiple analysis.

Discussion

Coronary restenosis. Conventional balloon angioplasty continues to be plagued by a relatively high (30% to 40%) incidence of restenosis (1–3) and certain patient or lesion subgroups (prior restenosis, "type B lesion" [for example, eccentric or ostial], long lesions, total occlusion or vein graft stenoses) appear to have restenosis rates approaching 50% (13). No alteration in dilation technique or concomitant medical therapy has yet been shown conclusively to reduce the incidence of restenosis (14).

The observed rate of restenosis may vary, depending on the patient or lesion groups treated, as well as on which definition of restenosis is employed (15,16). *Clinical restenosis*, defined as recurrent angina or exercise test evidence of ischemia, is neither sufficiently sensitive nor specific for rigorous analysis and may be confounded by the presence of collateral flow, prior infarction or other diseased vessels (17). Even *angiographic restenosis* may be misleading when the restudy rate is <75% because disproportionate follow-up data may lead to selection bias (18). Moreover, the observed angiographic restenosis rate may vary, depending on

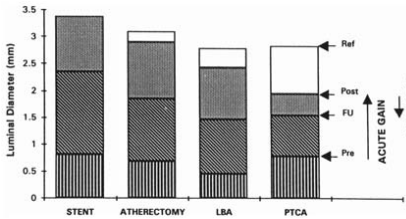

Figure 3. Stacked bar graphs compare the average lumen diameter before intervention (Pre), after intervention (Post) and at follow-up study (FU) with reference artery size (Ref) among the three devices and conventional angioplasty (PTCA, Nobuyoshi et al. [23]). Acute gain is defined as the immediate improvement in lumen diameter provided by the intervention, whereas late loss is the subsequent reduction in lumen diameter at 6-month follow-up study. (The reference diameter is not displayed for stents because the average postprocedure diameter was actually larger than the reference diameter.) Late loss (**stippled bar**) is seen to be equal among the three interventions and twice as great as that seen after conventional angioplasty. LBA = laser balloon angioplasty.

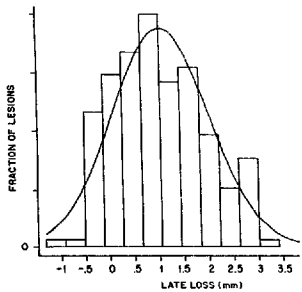

Figure 4. Histogram distribution of late loss in 184 lesions restudied at 6 months after treatment with the new devices. All three interventions are pooled given their equivalent late loss. The normal distribution of late loss is apparent.

whether visual or measured stenosis severity estimates are used (19) and which definition of restenosis is applied. In common use are the National Heart, Lung, and Blood Institute IV definition (loss of 50% of the acute gain produced by the procedure) (16,20) or the Thoraxcenter definition (loss of 0.72 mm of absolute lumen diameter after the procedure [twice the standard deviation for repeat determinations of arterial diameter obtained with their measurement system]) (21).

Because new devices generally produce better acute results than does conventional angioplasty, both of the commonly used angiographic definitions (16,20,21) may identify some patients as having restenosis, who have comparatively mild (<50% stenosis) lesions at the time of follow-up angiography. Therefore, some centers have preferred to define restenosis as a late diameter stenosis ≥50% relative to the reference segment (22). In our analysis, we sought to avoid these pitfalls by obtaining a high (83%) level of angiographic follow-up data, using digital caliper measurements of optically magnified images and developing a novel ("definition-independent") graphic method for displaying the late behavior of different groups of patients (Fig. 2). This graphic analysis (and application of the traditional dichotomous definition of >50% stenosis) shows that single stents are associated with a lower incidence of restenosis than is either atherectomy or laser balloon angioplasty in this nonrandomized series of patients.

Differences in restenosis with the three devices. In part, the observed differences in restenosis may relate to differences in the patients treated with individual devices. For example, laser balloon angioplasty was performed solely on new stenoses, whereas the majority of stents were placed in patients who had prior restenosis. Although all patients had focal disease of arteries >2.5 mm and the majority had at least one unfavorable characteristic for subsequent restenosis (prior restenosis, eccentricity, saphenous graft location), other disparate patient characteristics preclude conclusive comparisons of device-specific restenosis rates. They do not, however, compromise the analysis of late lumen diameter in terms of acute gain and late loss given the uniform behavior of the three devices in this model.

Late loss in lumen diameter with the three devices. The central question then becomes whether the apparent difference in the incidence of restenosis among devices was due to differences in the amount of late loss. A number of postmortem and in vivo atherectomy analyses (9,23,24) have confirmed that intimal hyperplasia (smooth muscle cell migration, proliferation and matrix synthesis) is the major contributor to the progressive lumen reduction that takes place after successful dilation. Some such reduction in lumen diameter has been noted in virtually all patients restudied after angioplasty (2,13). Although no drug regimen has yet conclusively reduced restenosis, it was hoped that the new devices in question (stents, atherectomy and laser balloon angioplasty catheters) would provide a smoother lumen surface less stimulatory of subsequent intimal hyperplasia. If, however, the magnitude of this hyperplasia is reflected by the late loss in lumen diameter that takes place between the posttreatment and 6-month follow-up angiograms, it is clear that each of the three new technologies studied actually triggered *more* average late loss (1 mm) than has typically been seen after conventional balloon angioplasty (0.4 mm) (1,2). This outcome should not be unexpected given greater vessel deformation with the new devices and the potential mitogenic stimulus resulting from implantation of a metallic stent, exposure of deep wall components by atherectomy or thermal denaturation of proteins by laser balloon angioplasty.

Acute gain in lumen diameter with the three devices. If the observed differences in restenosis rates seen among these devices were not due to differences in late loss, they must have been the result of the differences in acute gain (1.7 to 2.7 mm). The acute gain with conventional angioplasty is generally limited to 1.2 mm, leaving an average residual stenosis of 30% after successful conventional angioplasty (1,13). This limitation is primarily due to pronounced elastic recoil of the vessel after balloon deflation (25), which may negate >1 mm of the potential lumen diameter present during full balloon inflation. Attempts to overcome this limitation by the use of larger balloon diameters (balloon/artery ratio >1.1:1) tend to increase dissection and abrupt vessel closure (26).

In contrast, all three newer devices achieved greater acute gain than does conventional angioplasty, resulting in a very low postprocedure percent stenosis: −1% for stents, 6% for atherectomy and 15% for laser balloon angioplasty. This improvement in acute gain may be due to reduction in elastic recoil by metallic stenting and laser balloon welding or to plaque removal by directional atherectomy (27). Moreover, these devices appear to be able to achieve their larger acute gains without a corresponding increase in the inci-

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dence of major complications (7,9,10). When the resultant acute gain is large enough (as is the 2.7-mm gain with stent placement), it appears to be able to offset the 1-mm average late loss, while still maintaining a large lumen diameter and a favorably low (19%) overall restenosis rate at follow-up study. In contrast, smaller acute gains coupled with an equivalent 1-mm late loss (Table 1) are reflected in correspondingly higher rates of restenosis seen after atherectomy (31%) and laser balloon angioplasty (50%) in this study.

Implications. These observations have several important implications for coronary intervention. First, there is still no evidence that any of these new devices decrease the amount of subsequent intimal hyperplasia observed with conventional balloon angioplasty (in fact, they all seem to increase intimal hyperplasia). Second, the ability of a device to reduce restenosis appears to depend on its producing sufficient immediate increase in lumen diameter (acute gain) to offset the expected subsequent intimal hyperplasia (late loss). These observations may prompt operators to achieve the largest acute gains that can be obtained safely using their particular new technology. Third, the persistent occurrence of restenosis in 19% of stented lesions (which had an average −1% residual stenosis immediately after stenting), suggests that no device will be able to further reduce the restenosis rate unless it (or its concomitant pharmacotherapy) also reduces the amount of late loss. Finally, the observation that late loss is a ubiquitous and normally distributed phenomenon suggests that we should begin to approach restenosis as a continuous rather than a binary (restenosis/no restenosis) outcome. That approach may prove particularly valuable in the analysis of future comparisons among devices, patient categories or drug treatment regimens.

Limitations. This study has several potential limitations. First, the use of measured late loss as a marker for the amount of intimal hyperplasia has not been validated directly, although we have confirmed (9) the presence of intimal hyperplasia during atherectomy of restenoses occurring after each type of the studied interventions. Furthermore, although late loss after angioplasty may be the summation of intimal hyperplasia and elastic recoil, we disregarded the contribution of the latter in our analysis of these new devices. After stenting, we confirmed that measured late loss correlates well with direct measurement of the intrastent filling defect seen at follow-up angiography (7), and after laser balloon angioplasty, elastic recoil does not appear to be significant because lumen diameters were equivalent between immediate postprocedure and 24-h restudy (10). Although elastic recoil after atherectomy has not been evaluated precisely, lumen enlargement is only partially due to dilation (27), and interruption of elastic components by subintimal vessel wall excision may be common (9).

Second, measurement of late loss at 6 months assumes that all treated segments have reached their maximal narrowing by that time. Comparison of 6- and 12-month restudies in patients receiving a stent has demonstrated no further reduction in lumen diameter after 6 months (Schatz, personal communication), a finding that parallels prior observations (2,13) in consecutively studied patients undergoing angioplasty, showing that intimal hyperplasia reaches its final thickness by 4 months. Earlier (<4-month) restudy of seven symptomatic patients with restenosis in our study may have truncated their potential late loss by the premature development of clinical symptoms that triggered further intervention. Intravenous nitrates were given to all patients during their acute study only, but analysis showed no difference in reference vessel diameter between the acute and follow-up angiograms.

Third, the observed normal distribution of late loss and the similarities in mean late loss among the three treatments rest on the gross clinical similarities among the individual patients treated. Pending completion of randomized trials, a multivariable model could be developed to correct for characteristics found to be significant determinants of late loss to reduce the effect of the evident interdevice patient selection biases. Such a model would require a much larger sample size but is being pursued using multicenter data for conventional angioplasty and newer devices. These differences in lesion characteristics, however, compromise neither the across-device application of our model (based on acute gain and late loss) nor the conclusion that differences in restenosis rates relate to differences in acute gain. In fact, the device with the lowest apparent restenotic rate (the stent) had the highest percent of prior restenotic lesions, whereas the device with the highest apparent restenosis rate (laser balloon angioplasty) was used only in new lesions.

Conclusions. Detailed 6-month angiographic follow-up in a cohort of patients treated with one of three investigational technologies for coronary intervention at a single center suggest a lower (19%) restenosis rate for stents compared with rates obtained with directional atherectomy (31%) or laser balloon angioplasty (50%). These differences in apparent restenosis rate do not seem to be the result of less intimal hyperplasia (late loss) in those with a stent because late loss was similar (1 mm) for all three interventions, amounting to more than twice that reported for conventional angioplasty. Rather, the lower observed restenosis rate for stenting appears to be the direct result of the larger posttreatment lumen diameter achieved by stenting, which allows tolerance of late loss in most patients without the generation of angiographic restenosis (late diameter stenosis ≥50%). This approach to the analysis of restenosis may prove useful in subsequent trials comparing devices, patient groups or drug regimens. The observed lower restenosis rate in stented lesions, however, will need to be confirmed by randomized trials now underway.

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